The associations of triclosan and paraben exposure with allergen sensitization and wheeze in children

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ABSTRACT

Triclosan and parabens are chemicals used in personal care and medical products as microbicides and preservatives. Triclosan and paraben exposure may be associated with allergy (atopy), but these associations have not been evaluated with respect to other atopic states such as eczema (atopic dermatitis). This study examines the associations of urinary triclosan and paraben concentrations with allergic sensitization and asthma in children according to eczema history. We performed a cross-sectional analysis of U.S. children aged 6–18 years who participated in the National Health and Nutrition Examination Survey (2005–2006). Triclosan and paraben concentrations were measured in urine. We assessed associations of triclosan and parabens with allergic sensitization and asthma using multivariable logistic regression in 837 children with complete data and stratified our results by eczema status. After covariate adjustment, triclosan and methyl and propyl paraben concentrations were positively associated with the odds of aeroallergen sensitization. Eczema did not significantly modify the association between triclosan or paraben levels and aeroallergen sensitization, asthma, or wheeze. The odds of parent-reported atopic asthma increased 34% (95% CI, 0, 81) across triclosan concentration quartiles. Increasing triclosan concentrations (quartiles) were associated with 2.3 times the odds of food sensitization (95% CI, 1.14, 4.44) among children with eczema, but not among children without eczema (OR, 1.25; 95% CI 0.93, 1.68; effect measure modification, $p = 0.04$). Triclosan and paraben exposures may increase the risk of atopic asthma and aeroallergen sensitization. Prospective studies are necessary to confirm these findings and determine if these chemicals pose a risk to children's health.

(Allergy Asthma Proc 35:475–481, 2014; doi: 10.2500/aap.2014.35.3803)

Child allergy and asthma prevalence have risen over the past decades.[1–3] Numerous studies have evaluated potential risk factors for this increase, but the reasons remain unclear.[4,5] Novel environmental exposures may partially explain the rising prevalence, but the waxing and waning of allergy and asthma symptoms in children makes investigation of causal factors challenging.

Several prospective studies provide evidence for an allergic progression in children, referred to as the "atopic march."[6–11] The atopic march describes the observed natural progression of clinical signs of allergic/atopic disease from atopic dermatitis (AD) or eczema to allergic rhinitis and asthma and the waxing and waning of these symptoms.[6,10–12] It has also been shown that some children with a history of early AD are at higher risk of asthma.[7,10] Thus, in studies of risk factors for allergic rhinitis and asthma it may be important to consider history of AD because it is an early marker of the initiation of the atopic march or susceptibility to atopic disease.

Triclosan is a chemical that has been added to many personal care and medical products including toothpaste and soaps for its antimicrobial properties.[13,14] Parabens are the alkyl esters of *p*-hydroxybenzoic acid and are added to food, pharmaceuticals, and personal care products as preservatives because of their antimicrobial properties.[15] Investigators have noted that triclosan and parabens have endocrine disrupting properties and possibly immune modulating properties.[13,15–17] Three recent studies of triclosan and parabens established an association of exposure with allergy or hay fever diagnosis.[16–18] The mechanism for this association remains unclear, and none of these studies evaluated the relationship in the context of the atopic march/eczema history.

The objective of this study was to examine the association between urinary triclosan and paraben concentrations with allergic sensitization and asthma in 6- to 18-year-old U.S. children according to child eczema status using data from the National Health and Nutrition Examination Survey (NHANES).

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Presented (an earlier version of this project) at the Pediatric Academic Societies Meeting, Vancouver, BC, Canada, May 4, 2014

The authors have no conflicts of interest to declare pertaining to this article

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PARTICIPANTS AND METHODS

Data Source

We obtained data from the 2005–2006 NHANES, a nationally representative sample of the U.S. population. Approximately 5000 persons are surveyed biannually. The survey component includes demographic, socioeconomic, and health questions, while the examination component consists of medical, dental, physiological, and biospecimen measurements. We included children aged 6–18 years because urinary triclosan and paraben concentrations were not determined in children <6 years old. The Penn State University Hershey Medical Center Institutional Review Board reviewed this project.

Measurement of Urinary Triclosan and Parabens

Urinary concentrations of triclosan and parabens (butyl, ethyl, methyl, and propyl) were measured in spot urine specimens from a random, one-third subsample of participants >6 years of age and quantified using solid-phase extraction coupled to high-performance liquid chromatography and tandem mass spectrometry.[19–21] The limits of detection (LOD) in 100 μL of urine were 0.1–2 ng/mL.[19] We evaluated the distribution of the chemicals and determined that many were right skewed and some had a high proportion of values below the LOD. Using these distributions as a guide, we divided triclosan into quartiles, log-transformed propyl and methyl paraben, and dichotomized butyl and ethyl paraben (at LOD) for analysis. We included urinary creatinine as a covariate in all analyses to adjust for urinary dilution.[20,21]

Measures of Allergic Sensitization

Children >6 years had a full allergen-specific sensitization evaluation panel. ImmunoCap (Uppsala, Sweden) was used to determine allergen specific sensitization to *Dermatophagoides farinae*, *Dermatophagoides pteronyssinus*, cats, dogs, cockroaches, *Alternaria*, peanuts, eggs, milk, ragweed, rye grass, Bermuda grass, oak, birch, shrimp, *Aspergillus*, thistle, mice, and rats. We considered a participant to be sensitized to an allergen if their serum-specific allergen IgE level was ≥0.35 kU/L. We categorized allergens into food (peanut, egg, milk, and shrimp) or aeroallergen (all others) and considered a child sensitized/atopic if they were sensitized to any allergen in that category.

Measures of Asthma or Wheeze

We defined a child as having doctor-diagnosed asthma if the response was yes to the question, "has a doctor or other health professional ever told [you/study participant] that [you have/study participant has] asthma?" We defined a child as having wheeze in the past 12 months if the response was yes to the question, "in the past 12 months have [you/study participant] had wheezing or whistling in [your/study participant's] chest?" We considered a child atopic if the child had at least one sensitization (≥0.35 kU/L) to any allergen, and we used this definition to categorize children into atopic or nonatopic asthma and atopic or nonatopic wheeze groupings.

Eczema and Potential Modifiers or Covariates

We defined a child as having eczema if the response was yes to the question, "has a doctor or other health professional ever told [you/study participant] that [you have/study participant has] eczema?" We chose, *a priori*, four variables to include as covariates: race, poverty income ratio, age, and sex. We grouped race/ethnicity into Hispanic, non-Hispanic white, non-Hispanic black, and other. We grouped the poverty–income ratio variable dichotomously <1 or ≥1. We categorized age into 6–11 years and 12–18 years, to emulate NHANES prevalence reports.

Statistical Analysis

We calculated descriptive statistics for all demographic, exposure, and outcome data. The survey sampling design was accounted using the PROC RLOGIST and PROC MULTILOG procedures in SUDAAN 11.0.0, with the default method for calculating robust standard errors.[22] A two-sided, 5% level to test statistical significance was used.

Logistic regression analyses were used to examine the bivariate association of triclosan and parabens with allergic sensitization and eczema, and multinomial logistic regression analyses to examine the bivariate association of triclosan and parabens with atopic and nonatopic asthma and wheeze. The models were then repeated including the selected covariates assumed to be candidate confounders of the relationship between exposure and outcome. We report odds ratios and multinomial odds ratios (representing a change across more than one outcome group). Because we were specifically interested in the atopic march and eczema is an early step in the atopic march, we assessed effect measure modification by testing the interactions between eczema and triclosan or parabens in each of these multivariable models.

RESULTS

Of 3165 children aged 6–18 years, 1019 participants had urinary triclosan and paraben measurements and 837 of these had complete outcome data with nonmissing covariates. The weighted mean age was 12.3 years (95% CI, 12.0, 12.6) and 52.1% were boys (Table 1). The concentration of triclosan, methyl paraben, and propyl

Table 1 **Study participant characteristics (n = 837)**

	n	%	Weighted (%)
Sex			
Male	438	52.3	52.1
Female	399	47.7	47.9
Race/ethnicity			
Hispanic	300	35.8	16.2
Non-Hispanic white	238	28.4	62.2
Non-Hispanic black	249	29.8	14.3
Other	50	6.0	7.4
Poverty–income ratio			
<1	236	28.2	19.1
≥1	601	71.8	80.9
Age			
6–11 yr	287	34.3	42.9
12–18 yr	550	65.7	57.2

paraben was detectable in the majority of children (Table 2). The mean concentration of butyl paraben was the lowest of the reported parabens, whereas methyl paraben was highest.

Aeroallergen sensitization was present among 42.8% of participants and food sensitization was present among 19.2% of the participants. The prevalence of asthma diagnosis, wheeze, and eczema was 14.4, 11, and 11.7%, respectively (Table 3).

Bivariate Associations of Urinary Triclosan and Parabens and Covariates with Outcomes

There was an association of sex, race, triclosan, methyl paraben, and propyl paraben with aeroallergen sensitization (Table 3). Race was associated with food sensitization; non-Hispanic black race had the highest frequency of food sensitization. Triclosan, propyl paraben, and eczema were associated with asthma; eczema was associated with atopic asthma, having higher propyl paraben concentrations was associated with increased frequency of nonatopic asthma, and having higher triclosan concentrations was associated with decreased frequency of nonatopic asthma. Race, triclosan, and eczema were associated with wheeze; non-Hispanic black race was associated with increased frequency of atopic wheeze, having higher triclosan concentrations was associated with increased frequency of atopic wheeze and decreased frequency of nonatopic wheeze, and having eczema was associated with increased frequency of atopic wheeze. Poverty–income ratio and age were associated with eczema; a higher poverty–income ratio was associated with increased frequency of eczema and lower age was associated with increased frequency of eczema.

Multivariable Association of Urinary Triclosan and Parabens with Outcomes

In multivariable analysis, adjusting for urinary creatinine, age, poverty–income ratio, sex, and race, we found that increasing urinary triclosan, methyl paraben, and propyl paraben concentrations were associated with increased odds of aeroallergen sensitization (Table 4), although there was no significant dose–response association. Triclosan and parabens were not associated with food sensitization or eczema.

In multivariable analysis, adjusting for urinary creatinine, age, poverty–income ratio, sex, race, and eczema, being in the highest quartile of triclosan exposure (compared with the lowest quartile) was associated with 2.5 times the odds (95% CI, 1.03, 5.92) of atopic asthma (Table 5). There was a monotonic dose–response association of urinary triclosan concentration and odds of atopic asthma. Parabens were not associated with atopic asthma, and triclosan and parabens were not associated with nonatopic asthma.

In multivariable analysis, triclosan and parabens were not associated with atopic wheeze. Compared with the lowest quartile of triclosan exposure, being in the second or third quartile of triclosan exposure was associated with decreased odds of having nonatopic wheeze, respectively (Table 5). Increased methyl paraben exposure was also associated with decreased odds of nonatopic wheeze.

Interaction of Triclosan and Paraben Concentrations with Eczema

There was no significant modification of the association between urinary triclosan or paraben concentrations with aeroallergen sensitization, asthma, or wheeze according to eczema status. The association between urinary triclosan concentrations and food sensitization was significantly modified by eczema status (Table 6; $p = 0.04$). Increasing urinary triclosan concentrations were associated with increased odds of food sensitization among children with eczema; however, these associations were very imprecise, as evidenced by the very wide 95% CIs, the small number of exposed children with eczema, and food sensitization ($n = 19$; 9.5%) compared with children without eczema and higher triclosan concentrations (Table 6).

DISCUSSION

We found that detectable urinary triclosan and methyl and propyl paraben concentrations were common and were associated with increased odds of aeroallergen sensitization in a nationally representative sample of 6- to 18-year-old children. Triclosan was associated with food sensitization, but this association was stronger among children with eczema, suggesting that the associations may be stronger among children with an atopic state.

Table 2 **Study population exposures (n = 837)**

	n	%	%*	Geometric Mean (95% CI)*
Triclosan (ng/mL)				15.5 (13.0, 18.6)
Q1 (<3.8)	209	25.0	25.0	
Q2 (3.8–12.14)	210	25.1	24.7	
Q3 (12.15–53.99)	209	25.0	26.6	
Q4 (≥54.0)	209	25.0	23.7	
Butyl paraben (ng/mL)				0.4 (0.33, 0.48)
<0.20	436	52.1	54.36	
≥0.20	401	47.9	45.64	
Ethyl paraben (ng/mL)				1.29 (1.14, 1.47)
<1.00	573	68.5	73.5	
≥1.00	264	31.5	26.5	
Methyl paraben (ng/mL)				42.0 (35.2, 50.2)
<34.0 (median)	323	38.6	50.7	
≥34.0	514	61.4	49.3	
Propyl paraben (ng/mL)				5.3 (4.0, 7.0)
<4.3 (median)	345	41.2	49.9	
≥4.3	492	58.8	50.1	

Survey weighted results.

Triclosan was also associated with increased odds of atopic asthma. These exposures likely originate from consumer products; therefore, the associations may have important implications across populations.

Mean triclosan and paraben levels were similar to those reported in other studies of U.S. children,[23–25] but triclosan was more frequently detected in these U.S. children than in Norwegian children.[18] Because these chemicals are nonpersistent in the body, urine concentrations are reflective of exposure at a single time. Prior studies suggest that repeated urinary paraben and triclosan concentrations are only modestly correlated, suggesting the potential for exposure misclassification.[25,26] In large enough sample sizes and assuming nondifferential exposure misclassification, a single urinary measure of triclosan or paraben might provide adequate representation of an individual's recent exposure. However, future studies should investigate exposures at earlier life stages, particularly before the development of allergic symptoms.

Several investigators have noted triclosan and parabens have immune modulating properties in cross-sectional analysis. Clayton *et al.* evaluated NHANES 2003–2006 and reported an association of higher triclosan levels and allergy or hay fever diagnosis.[16] Savage *et al.*, evaluated data from NHANES 2005–2006 and described an association of triclosan and paraben with increased odds of aeroallergen and food sensitization.[17] Bertelsen noted that triclosan concentrations were associated with aeroallergen, rather than food sensitization among Norwegian children.[18] Although these three recent studies evaluated triclosan and parabens, none reported a consideration of other key covariates that might be part of the atopic march, such as diagnosis of eczema. Our findings of an association of triclosan and paraben exposure with increased odds of aeroallergen sensitization replicate that of Savage *et al.*[17] However, we extend the evaluation by including eczema in the analysis. Although eczema did not modify the associations of triclosan and aeroallergen sensitization, it affected the food sensitization association. Triclosan was associated with food sensitization, but this association was stronger among children with eczema.

The mechanism for the stronger association of triclosan with food sensitization among children with eczema is unclear. The presence of the statistical interaction is consistent with effect measure modification on the multiplicative scale. This could be an additional finding of evidence for an atopic march in children, which is difficult to disentangle in a cross-sectional analysis.[6–9] The association could also be related to differential susceptibility of children with eczema. We can not completely discount the possibility of reverse causality in this association—triclosan-containing products may be used more by children with eczema, but this is not likely to explain the relationship because there was a significant dose–response association of triclosan with food sensitization among these children with eczema and the mean triclosan concentration was lower among children with eczema (data not shown). Recent hypotheses about the development of food sensitization suggest that exposure *via* the skin may lead to allergic sensitization, and some investigators suggest that eczema may play an important role in the development of food sensitiza-

Table 3 **Survey weighted, unadjusted association of the participant characteristics and exposures with atopic outcomes (n = 837)**

Characteristic or Exposure	Aeroallergen Sensitization n = 834		Food Sensitization n = 833		Asthma n = 837		Wheeze n = 837		Eczema n = 837	
	Yes	No	Yes	No	Atopic	Nonatopic	Atopic	Nonatopic	Yes	No
Total population	42.8%	57.2%	19.2%	80.8%	9.2%	5.2%	7.2%	3.8%	11.7%	88.3%
Sex	**	**								
Male	50.1	49.9	22.0	78.0	10.6	5.9	10.3	2.7	13.3	86.7
Female	34.8	65.2	16.2	83.8	7.6	4.5	3.9	4.9	9.9	90.1
Race/ethnicity	*	*	***	***			*			
Hispanic	41.8	58.3	14.3	85.8	8.3	9.5	4.2	7.0	5.4	94.6
Non-Hispanic white	39.8	60.2	15.8	84.3	9.0	4.0	7.4	3.3	12.4	87.6
Non-Hispanic black	57.8	42.2	34.9	65.1	10.5	6.1	9.2	2.1	16.2	83.8
Other	41.6	58.4	29.0	71.0	10.1	4.5	7.8	3.8	10.7	89.3
Povertyincome ratio									**	**
<1	43.3	56.7	23.2	76.8	9.3	7.0	6.1	4.0	4.6	95.4
≥1	42.7	57.3	18.3	81.7	9.1	4.8	7.5	3.7	13.4	86.6
Age (yr)									***	***
6–11	36.9	63.1	18.9	81.1	9.1	4.4	7.4	2.6	17.1	82.9
12–19	47.2	52.8	19.4	80.6	9.2	5.8	7.0	4.6	7.7	92.3
Triclosan	*	*				**	**	*		
Q1 (<3.8)	32.3	67.7	13.5	86.6	5.9	10.8	4.7	7.8	13.4	86.7
Q2 (3.8–12.14)	45.8	54.2	16.1	83.9	8.3	2.6	4.9	2.6	13.2	86.8
Q3 (12.15–53.99)	44.8	55.2	20.0	80.0	9.5	4.7	8.9	2.1	10.5	89.5
Q4 (≥54.0)	48.2	51.8	27.6	72.4	13.1	2.7	10.3	2.7	9.8	90.2
Butyl paraben										
≥0.20	46.2	53.8	20.7	79.3	7.4	3.3	7.5	3.1	13.5	86.5
<0.20	40.0	60.0	18.0	82.0	10.7	6.8	6.9	4.4	10.2	89.8
Ethyl paraben										
≥1.00	45.1	54.9	21.0	79.0	9.2	3.3	5.6	2.3	10.8	89.2
<1.00	42.0	58.0	18.6	81.4	9.2	5.9	7.8	4.3	12.0	88.0
Methyl paraben	*	*								
≥ median	47.3	52.7	20.3	79.7	10.8	3.8	8.3	3.0	14.1	85.9
< median	38.4	61.6	18.2	81.8	7.6	6.7	6.1	4.5	9.4	90.6
Propyl paraben	*	*				*				
≥ median	46.5	53.5	20.3	79.7	9.5	3.4	6.6	3.0	12.3	87.7
< median	39.1	60.9	18.2	81.8	8.8	7.0	7.8	4.5	11.0	89.0
Eczema					**		***			
Yes	56.2	43.8	26.1	73.9	21.5	10.9	19.8	5.5	—	—
No	41.0	59.0	18.3	81.7	7.5	4.5	5.5	3.5		

Note: Tests of significance were based on Wald tests examining hypothesis of null parameter estimates in bivariate survey weighted logistic/multinomial models.
*$p < 0.05$; **$p < 0.01$; ***$p < 0.001$.*

tion.[27,28] Our findings offer support of this hypothesis but prospective studies are needed.

We also found an association of triclosan exposure and atopic asthma. This association is noted as borderline by Savage, but that may be related to their analytic approach. Recently, in another publication, Savage reported that using NHANES 2005–2010 data, they found an association of urinary triclosan and an increased likelihood of reporting an asthma attack.[29] Our findings represent another example of cross-sectional evidence for the possible association of triclosan exposure and asthma.

There are several limitations to this study. First, the analysis was cross-sectional so these associations can not verify causality. Second, although the analytic sample is a large, representative population, urinary triclosan and paraben concentrations and allergy outcome data were

Table 4 **Adjusted associations of exposures with atopic outcomes, logistic regression results (n =837)***

Characteristic or Exposure	Aeroallergen Sensitization		Food Sensitization		Eczema	
	n (%)	OR (95% CI)	*n* (%)	OR (95% CI)	*n* (%)	OR (95% CI)
Triclosan						
Q1 (<3.8)	207 (24.8)	REF	#	#	209 (25.0)	REF
Q2 (3.8–12.14)	209 (25.1)	1.84 (1.16, 2.92)			210 (25.1)	0.86 (0.32, 2.30)
Q3 (12.15–53.99)	209 (25.1)	1.70 (1.10, 2.62)			209 (25.0)	0.64 (0.21, 1.91)
Q4 (≥54.0)	209 (25.1)	1.91 (1.02, 3.57)			209 (25.0)	0.68 (0.29, 1.57)
Butyl paraben ≥0.20 vs <0.20	399 (47.8)	1.53 (0.98, 2.38)	400 (48.0)	1.15 (0.60, 2.20)	401 (47.9)	1.91 (0.81, 4.50)
Ethyl paraben ≥1.00 vs <1.00	263 (31.5)	1.09 (0.72, 1.65)	263 (31.6)	1.02 (0.59, 1.77)	264 (31.5)	1.03 (0.45, 2.36)
Methyl paraben One log unit change	—	1.15 (1.06, 1.26)	—	0.98 (0.85, 1.14)	—	1.14 (0.92, 1.41)
Propyl paraben One log unit change	—	1.18 (1.08, 1.29)	—	1.04 (0.94, 1.15)	—	1.13 (0.92, 1.38)

**Adjusted for urinary creatinine, age, poverty–income ratio, sex, and race.*
#See Table 6 for extended model.
OR = odds ratio; REF = quartile of reference for odds ratio calculation.

Table 5 **Adjusted associations of exposures with asthma and wheeze, multinomial regression results (n = 837)***

Characteristic or Exposure	Asthma				Wheeze			
	Atopic		Nonatopic		Atopic		Nonatopic	
	n (%)	OR Atopic vs None (95% CI)	*n* (%)	OR Nontopic vs None (95% CI)	*n* (%)	OR Atopic vs None (95% CI)	*n* (%)	OR Nonatopic vs None (95% CI)
Triclosan								
Q1 (<3.8)	18 (23.1)	REF	17 (40.5)	REF	14 (23.3)	REF	10 (37.0)	REF
Q2 (3.8–12.14)	18 (23.1)	1.36 (0.74, 2.48)	7 (16.7)	0.23 (0.08, 0.69)	11 (18.3)	0.96 (0.23, 4.04)	6 (22.2)	0.31 (0.11, 0.85)
Q3 (12.15–53.99)	16 (20.5)	1.74 (0.53, 5.67)	10 (23.8)	0.50 (0.19, 1.32)	20 (33.3)	1.88 (0.88, 4.01)	5 (18.5)	0.27 (0.08, 0.98)
Q4 (≥54.0)	26 (33.3)	2.47 (1.03, 5.92)	8 (19.1)	0.28 (0.06, 1.41)	15 (25.0)	2.16 (0.54, 8.58)	6 (22.2)	0.33 (0.08, 1.37)
Butyl paraben ≥0.20 vs <0.20	37 (47.4)	0.56 (0.29, 1.08)	15 (35.7)	0.41 (0.12, 1.33)	34 (56.7)	1.11 (0.57, 2.15)	12 (44.4)	0.43 (0.08, 2.31)
Ethyl paraben ≥1.00 vs <1.00	30 (38.5)	1.03 (0.62, 1.73)	8 (19.1)	0.56 (0.15, 2.04)	21 (35.0)	0.64 (0.27, 1.50)	6 (22.2)	0.33 (0.08, 1.40)
Methyl paraben One log unit change	—	1.11 (0.84, 1.47)	—	0.78 (0.59, 1.02)	—	1.16 (0.80, 1.67)	—	0.76 (0.57, 0.99)
Propyl paraben One log unit change	—	1.05 (0.91, 1.21)	—	0.77 (0.58, 1.03)	—	0.97 (0.76, 1.22)	—	0.78 (0.59, 1.02)

**Adjusted for urinary creatinine, age, poverty–income ratio, sex, and race.*
OR = odds ratio; REF = quartile of reference for odds ratio calculation.

not available for all participants. Third, urinary triclosan and paraben concentrations vary over time, so use of single biomarker measures of exposure could result in attenuated effect estimates if nondifferential exposure misclassification is present. Fourth, the asthma and eczema outcomes were based on parent report, which may be subject to misclassification; however, both outcomes were based on standard questions used in the asthma and allergy literature. Fifth, as previously noted, reverse causality in this analysis is a possibility. Sixth, there is a possibility that other environmental exposures could have confounded our observed associations.

Table 6 **Adjusted interaction of triclosan exposure with eczema in food sensitization***

Characteristic or Exposure	*n* (%)	OR (95% CI)
Triclosan eczema, yes		
Q1 (<3.8)	19 (2.2)	REF
Q2 (3.8–12.14)	24 (2.9)	1.60 (0.33, 7.85)
Q3 (12.15–53.99)	17 (2.0)	21.21 (2.70, 166.71)
Q4 (≥54.0)	19 (2.2)	8.17 (0.93, 71.59)
Triclosan/eczema, no		
Q1 (<3.8)	189 (22.7)	REF
Q2 (3.8–12.14)	184 (22.1)	1.12 (0.47, 2.68)
Q3 (12.15–53.99)	192 (23.0)	1.04 (0.44, 2.43)
Q4 (≥54.0)	190 (22.8)	2.03 (0.85, 4.85)

**Adjusted for urinary creatinine, age, poverty–income ratio, sex, and race. Interaction value of p = 0.04.*
OR = odds ratio; REF = quartile of reference for odds ratio calculation.

CONCLUSION

We found that triclosan and paraben exposures were common. Triclosan and paraben levels were cross-sectionally associated with aeroallergen sensitization, and eczema status modified associations between triclosan and food sensitization. Prospective studies with repeated biomarkers of triclosan and paraben exposure and child allergen sensitization are necessary to confirm these findings and determine if these chemicals pose a risk to children's health.

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